

BIOPHYSICAL EVALUATION OF THE
HUMAN VESTIBULAR SYSTEM

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ABSTRACT

The physical properties of the vestibular system are examined to determine an analytical model of the canalicular response to rotation in a linear acceleration field. The distension of the membranous canal duct is shown to produce a "roller pump" action of the endolymph when the canal is rotated about an axis orthogonal to the linear acceleration vector. Experiments to evaluate further the apparently non-linear low frequency response of the semicircular canals are presented and discussed.

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I. THE RESPONSE OF THE SEMICIRCULAR CANALS TO
ROTATION IN A LINEAR ACCELERATION FIELD

(Experiments and Analysis by Robert Steer)

Several of the experiments which have been devised to study man's vestibular reactions to unusual motion environments have elicited modes of response that cannot be accounted for by existing physical descriptions or analytic models of the vestibular system. Of particular importance are the results of recent experiments which show that rotation of subjects at a constant angular velocity about a horizontal longitudinal axis provokes continuous vestibular nystagmus and subjective sensation of rotation. Classical concepts of the vestibular sensors predict only a transient nystagmus response which decays to zero for this stimulus and for constant velocity rotation about any axis. Thus it has become necessary to re-examine these classical concepts to determine which receptors are responsible for this mode of stimulation.

The Flexible Semicircular Canals as a "Roller Pump"

From histological studies by Igarashi, the position of the membranous labyrinth relative to the bony labyrinth has been accurately measured. Observations of Engstrom indicate that the duct of the membranous canal is attached by fibrous connections along its outer periphery to the bony canal. However, the inner periphery is relatively unattached and flexible, and Money has shown for pigeons that the duct is denser than either the endolymph or perilymph that surrounds

it. Under the influence of pure angular acceleration about an axis in the direction of the gravity field, the duct flexibility and density will have no significant consequences. However, if the gravity field is not colinear with the axis of rotation, a component of it will act upon the mass unbalance and can produce a movement of endolymph within the duct.

A flexible duct immersed in and containing an incompressible fluid will be distended by the influence of a linear acceleration if its density is different from that of the fluid, and a density difference between the interior and exterior fluids will further accentuate this distension. As shown in Fig. 1, a flexible circular duct which is attached along its outer periphery to a rigid structure and is denser than the fluid surrounding it will have a constricted cross-sectional area where the acceleration field pushes it against its support, and it will be expanded where the acceleration pushes it away from its support.

Further, as illustrated in Fig. 1, if the linear acceleration vector \bar{a} is slowly rotated at a constant angular velocity w , the constriction will move along the outer periphery of the duct in phase with the rotation of the acceleration vector. The effect of the moving constriction is then to move or pump the fluid in the duct in the direction of rotation. This pumping action works against the viscosity and inertia of the fluid, and a high angular rotation rates the

fluid that is being pushed by the moving constriction cannot be displaced fast enough. It thereby builds up a pressure gradient which expands the duct back toward its original uniform shape. Thus, for high angular rotation rates of the linear acceleration vector, the mass of the fluids acts as a hydro-mechanical filter which reduces the duct constriction and, along with it, the pumping action of the flexible tube.

For a flexible tube with an elastic flow restraint such as the cupula of the semicircular canals, the fluid is initially pumped against and displaces the elastic restraint which then produces a pressure differential across the tube. A static equilibrium state is then reached where the displaced elastic restraint provides sufficient pressure feedback to inhibit further flow. Thus, for a constant velocity of rotation in a linear acceleration field, a constant cupula displacement can be maintained by this flexible roller pump action.

A Model for Canalicular Response to Rotating Linear Acceleration Stimulation

For constant rotation of the semicircular canals, the steady state displacement of the elastic cupula provides a pressure differential across the cupula which inhibits further flow. All of the fluid displaced by the moving constriction must therefore flow back through the constriction, and the pressure drop of this "back flow" must exactly balance that caused by the displaced cupula.

The average velocity $\bar{v}(\frac{A_c}{A})$ of the backflow is given by the equation

$$\bar{v}(\frac{A_c}{A}) = \frac{(A - A_c) W R}{A} \quad (1)$$

where

A = cross sectional area of the unrestricted portion of the duct

A_c = cross sectional area of the constricted portion of the duct

W = rotation rate of the linear acceleration vector

R = radius of the torus

The static pressure differential Δp which can maintain an average flow of endolymph can be calculated from fluid dynamic analysis to be:

$$\Delta p = \frac{2\pi R}{D_d} \bar{v}(\frac{A_c}{A}) \quad (2)$$

where

$$D_d = \frac{a^2 R g}{8\mu} \quad (3)$$

μ = viscosity of endolymph (in poise)

a = radius of canalicular duct (in cm)

g = gravitational constant, 980 cm/sec²

From equations (1) and (2) we find that the cupular pressure differential Δp that a rotating constriction can maintain is

$$\Delta p = \frac{16\pi\mu}{a^2 g} (1 - \frac{A_c}{A}) W R \quad (4)$$

Further, it can also be shown that a pressure differential Δp across the membranous duct produces a cupular response which is related to angular acceleration stimulation α by the equation

$$\Delta p = 2 \pi R \rho g \alpha \quad (5)$$

From equations (4) and (5) we obtain the relationship between the rate of angular rotation, the magnitude of the duct restriction, and the equivalent constant angular acceleration that would produce the same steady state cupular displacement.

$$\alpha_{\text{equiv}} = \frac{8 \mu W}{a^2 \rho g^2} \left(1 - \frac{A_C}{A}\right) \quad (6)$$

For the human semicircular canals where

$$a = 0.015 \text{ cm}$$

$$R = 0.3 \text{ cm}$$

$$\rho = 1 \text{ gm/cm}^3$$

$$\mu = 0.0085 \text{ poise}$$

$$g = 980 \text{ cm/sec}^2$$

$$\alpha_{\text{equiv}} = 0.3 \left(1 - \frac{A_C}{A}\right) W \text{ rad/sec} \quad (7)$$

To establish the applicability of the roller pump principle to the semicircular canal, the relative magnitudes of the bias component of slow phase nystagmus from rotation in a linear acceleration field and the steady state nystagmus stimulated by constant angular acceleration can now be

compared to determine how large a distension of the duct is necessary to produce a significant physiological response.

From the data of Guedry, a $6^\circ/\text{sec}$ bias component of vestibular nystagmus is noted for a 1 rad/sec rotation about a horizontal longitudinal axis. Several experiments have shown that such a $6^\circ/\text{sec}$ slow phase velocity would also result from a $0.6^\circ/\text{sec}^2$ or $0.01 \text{ rad}/\text{sec}^2$ constant angular acceleration. Solution of equation (7) for the value of $\frac{A_c}{A}$ when

$$W = 1 \text{ rad/sec}$$

$$\alpha_{\text{equiv}} = 0.01 \text{ rad/sec}^2$$

yields

$$\frac{A_c}{A} = .97 \tag{8}$$

This shows that a mere 3% constriction in duct area, or correspondingly, a 1.5% contraction of the radius of the membranous canalicular duct can produce sufficient roller pump action to account for the observed bias component of nystagmus which results from constant rotation at 1 rad/sec in a 1 g acceleration field.

The rotation rate W_r at which the roller pump action diminishes is determined by the elasticity and strength of the fibrous attachments of the duct, and is not readily calculable. However, this cut off frequency can be accounted for by adjoining to equation (6) a high frequency lag term to provide

for a diminished response at higher rates of rotation. From this we obtain:

$$\alpha_{\text{equiv}} = \frac{8\mu}{a^2 \rho g^2} \left(1 - \frac{A}{C}\right) \left[\frac{W}{\left(1 + \frac{W}{W_l}\right) \left(1 + \frac{W}{W_r}\right)} \right]$$

Rotating Linear Acceleration Stimulation Experiments

To investigate the variation of the bias and the amplitude of the sinusoidal component of vestibular nystagmus as a function of rotation rate, the M.I.T. Instrumentation Laboratory Precision Centrifuge with a rotating platform at a 32 ft. radius was fitted with the Man-Vehicle Control Laboratory rotating chair simulator and six experimental subjects were rotated at 5, 7.5, 10, 20, 30, and 40 rpm in a 0.3 g horizontal acceleration field. Nystagmus was measured with eyes open in the dark by use of a Biosystems, Inc. pulsed-infrared eye movement monitor.

From the nystagmus recordings the slow phase nystagmus velocities were calculated and plotted. In Fig. 2 is shown a sample nystagmus recording for each of the six rotation rates used, and in Fig. 3 is shown a sample of the slow phase velocities for each rotation rate. The results show a persistent sinusoidal component (utricularly sensed) at the period of rotation for all subjects at all rotation rates. For most subjects, a clear bias component (canalicularly sensed) is observed for 5 and 7.5 rpm and for some it still exists at 10 rpm; however, for 20, 30, and 40 rpm it is not observable in any of the subjects tested. The amplitude of

the sinusoidal component increases with increasing rates of rotation.

To compare the results with those of the horizontal rotation experiments it is necessary, even though the assumption of linearity is tenuous, to normalize the results of our experiments with respect to a one g gravity field. In Fig. 4a and b our normalized results are plotted along with those of Benson and Guedry. The model predicted bias component is also plotted in Fig. 4a for an assumed upper break frequency of $W_r = 7.5$ rpm.

It does appear that the predicted responses from the "roller pump" model are borne out by the data in that at low and high rotation rates the bias component is not observable, and there is a general shape of measured response that does conform to the predicted second order system. Further, the experimental data show that the upper break frequency which we were unable to calculate because of insufficient data is in the range from 5 to 10 rpm.

In summary, these experiments, which provide a slightly different vestibular stimulation than the "barbecue spit" experiments of Guedry and Benson or the "revolution without rotation" experiments of Money, further verify the hypothesis that rotation at a constant velocity in a linear acceleration field does provoke vestibular nystagmus. The results of the analysis presented here show that a duct area constriction of only 3% provides sufficient roller pump action to generate

the observed bias component of nystagmus. In addition, the upper cut off frequency W_r was found experimentally to be in the range between 7.5 and 10 rev/min.

II. LOW FREQUENCY RESPONSE OF THE SEMICIRCULAR CANALS

(Experiments and Analysis by Gerald Katz)

The dynamic properties of the semicircular canals are generally modelled as a torsion pendulum quantitatively described by a linear second order differential equation. This model is characterized by two time constants, the values of which are well known both for subjective response to angular accelerations and for objective response as measured by eye nystagmus. While the assumption of linearity is warranted for a wide range of frequencies, Hixon and Niven in 1962 found that nystagmus elicited at low frequencies (0.02 and 0.04 Hz) appears to be dependent upon the magnitude of peak angular acceleration.

To examine further this apparent nonlinearity of the semicircular canal response, a series of experiments was carried out to measure the subjective and objective response to angular sinusoidal accelerations of predominantly low frequencies (.01 Hz to .1 Hz).

The Experiment

Experiments were performed on four subjects. Each subject was seated in the cab of the Man-Vehicle Control Labora-

tory rotating chair (see MV-67-2), his head securely positioned above the center of rotation by means of a head rest and head strap. His torso was held in place by an aircraft type of shoulder harness and seat belt. Within easy reach of his right hand was a simple directional switch to be used by the subject to indicate the subjective feeling of rotation direction.

Eye movements relative to the skull were measured by a non-contact method based upon detection of the difference in reflected light from the sclera and the iris on both sides of one eye. A commercial model of an eye movement monitor with a linear range of $\pm 15^\circ$ and a resolution of 0.1° , mounted on glasses worn by the subject, was used for the entire series of experiments described in this section.

To comply with requirements for complete darkness in the cab (elimination of visual fixation point), the necessary illumination of the eye ball was achieved by an infrared light. Recordings of the cab position and eye movements were taken continuously and simultaneously during the experiments.

The matrix of frequency and peak amplitude of sinusoidal acceleration presented to each subject is listed below (Table 1). All of the data points except those at .01 Hz and .02 Hz were run for a length of time (several cycles) such that the results from ten cycles of readable data could be collected and averaged. The first sixty seconds of all records were disregarded to allow the transient response of the canals to

F (Hz)	α_m deg/sec	10	15	20	25	30	35	40
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.01		x	x					
.02				x	x	x	x	x
.04				x	x	x	x	x
.08				x	x	x	x	x
.10				x	x	x	x	x

Table 1. Matrix of Data Points Presented

attenuate. At .01 Hz and .02 Hz the peak angular velocity was great enough to cause discomfort for the subject if he was exposed to oscillation at these frequencies for any protracted length of time (greater than 5 minutes). We attempted to collect five or six cycles of readable data on two or three different runs at the same data point.

The subjective response of the subject to angular acceleration was indicated by means of the directional switch previously mentioned. Each subject impulsed the switch in the direction that he felt he was rotating. This directional impulse signal, the input sinusoid to the system, the angular position of the chair, and the nystagmus were all recorded simultaneously on a four channel pen recorder. The subjective switches and the transition point from left to right beating nystagmus could readily be compared to the actual zero angular velocity points of the chair.

Results and Discussion

No quantitative inferences were drawn, nor will any be reported, from the data gathered. It is felt that at least ten more hours of experimentation (in addition to the five already expended) per subject is necessary before there would be a sound statistical basis upon which to rest any quantitative conclusions.

Although any thought of deriving some quantitative knowledge from the experiments must be put aside, there is a wealth of qualitative understanding available. This encourages one about the possibility of gaining numerical understanding by expansion of the present experiments.

Plotting the subjective and objective phase shift results of these experiments against the theoretical description of the horizontal semicircular canals results in Figs. 5 and 6. In Fig. 5, most of the data falls below the theoretical curve. In Fig. 6, most is above the theoretical curve. This confirms the observation that the objective nystagmus response exhibits more phase lead than the subjective response at identical stimulus conditions. In the past, however, the two responses were elicited and recorded separately. They were recorded simultaneously in the present experiments. It is heartening to note that although there is a very wide spread, the locus of the center of gravity of the data for the subjective response does lie but slightly below the theoretical curve. Upon the collection of more data, a least squares fit could be made to determine whether there is a discrepancy.

Fig. 7 is a plot of the composite results of the present experiments against the Hixon and Niven composite results. There is a great deal of correlation noticeable here. Hixon and Niven suggested that there was an inherent nonlinearity in the vestibular system evident at low frequency-low angular acceleration stimulation which they proposed could be attributed to the cupula exceeding an angle of deflection that would still enable it to seal the ampulla. Leakage around the cupula by the endolymph would cause the nonlinear response. Although it cannot be concluded from the presented experiments that the nonlinearity is not present, it also cannot be shown that there is a linear relationship between phase lead and stimulus frequency regardless of amplitude of acceleration.

It is evident that our experimental data is not sufficient to support or negate the existence of low frequency nonlinearity for the semicircular canal model. Further experimentation to expand the scope of knowledge in this area is planned for the first half of 1968.

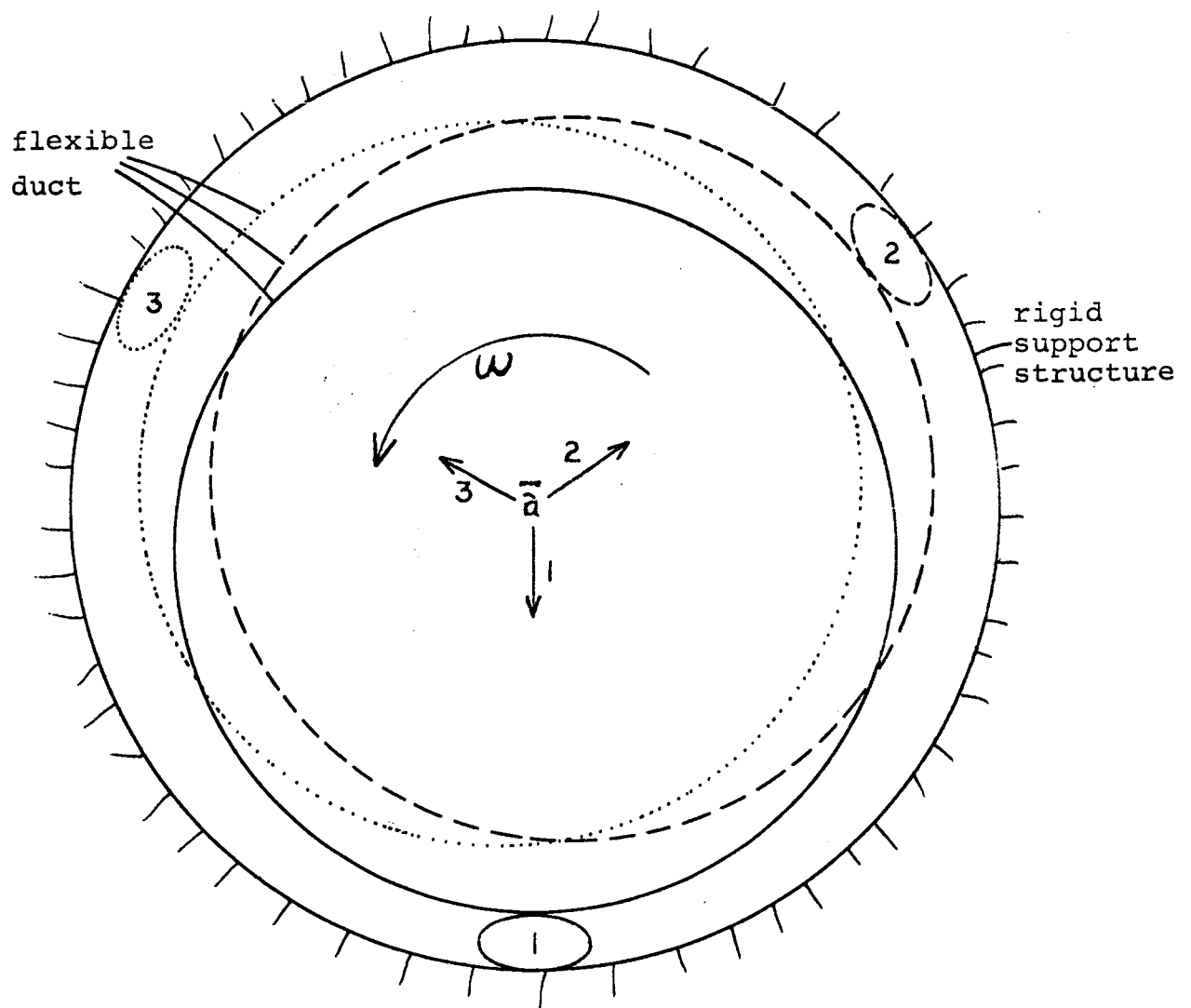


Figure 1

Illustration of the pumping action of the distended
duct when the linear acceleration field
is rotated at a uniform angular
velocity

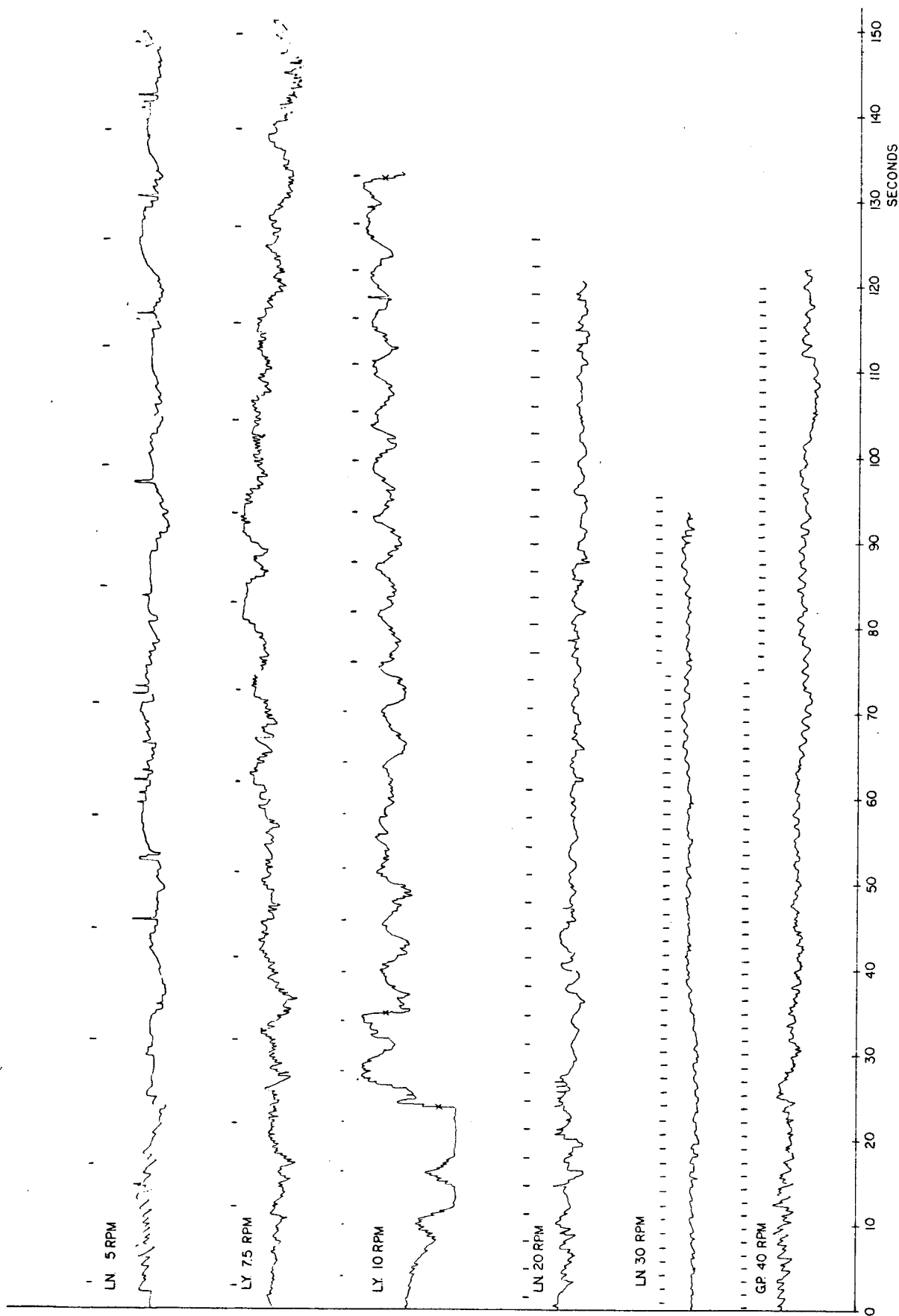


Figure 2

Sample eye movement recordings for rotations in 0.3 g lateral field

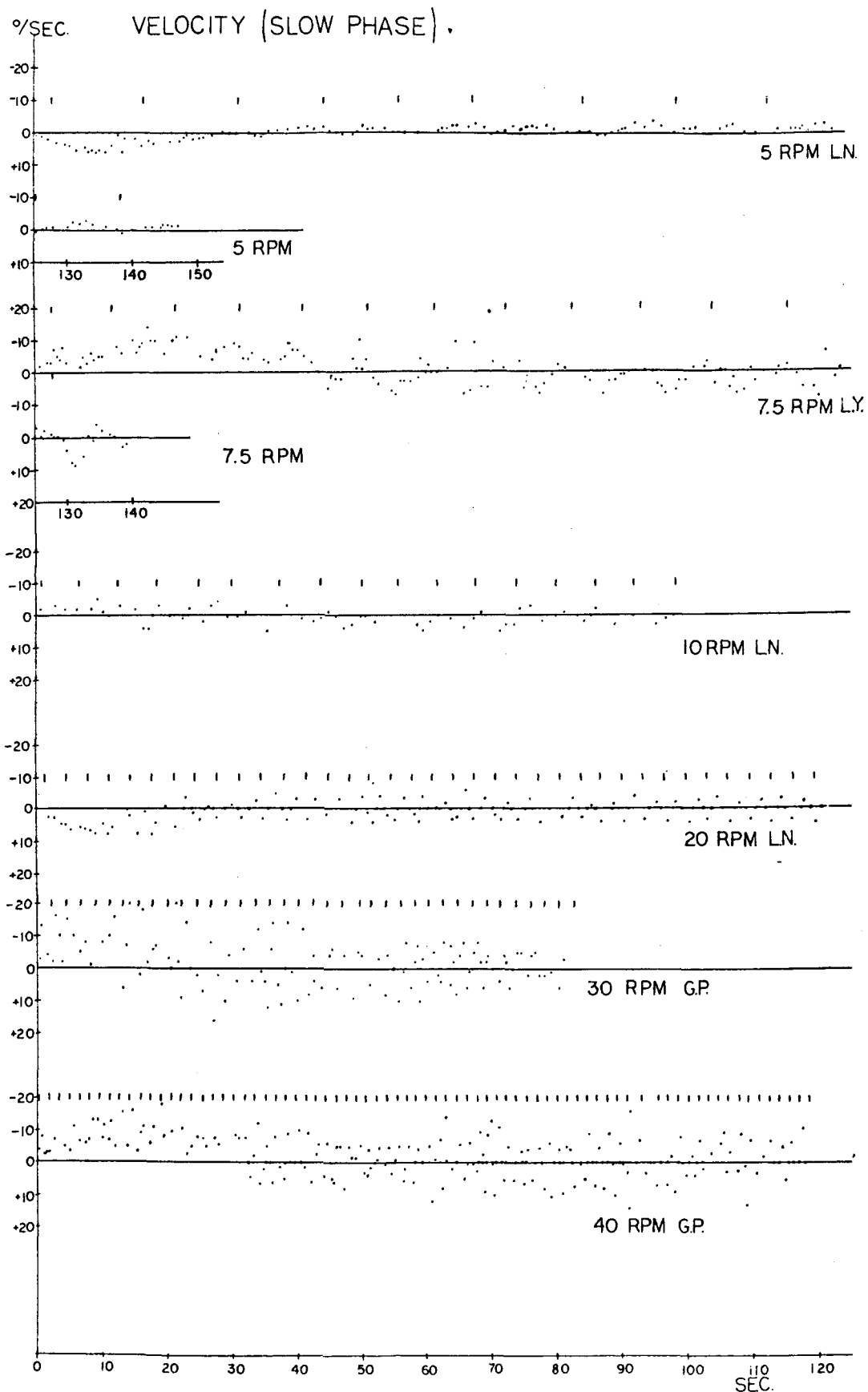


Figure 3
Slow phase nystagmus velocities from
rotation in 0.3 g field

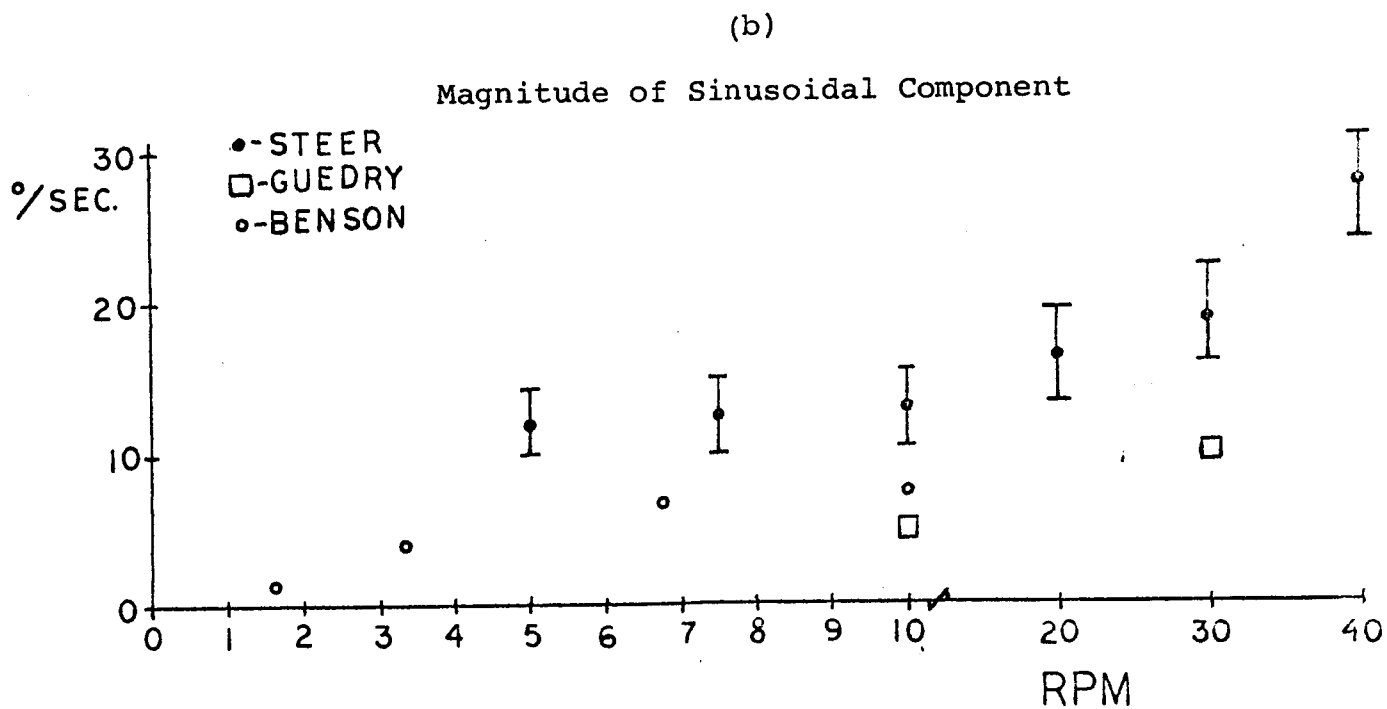
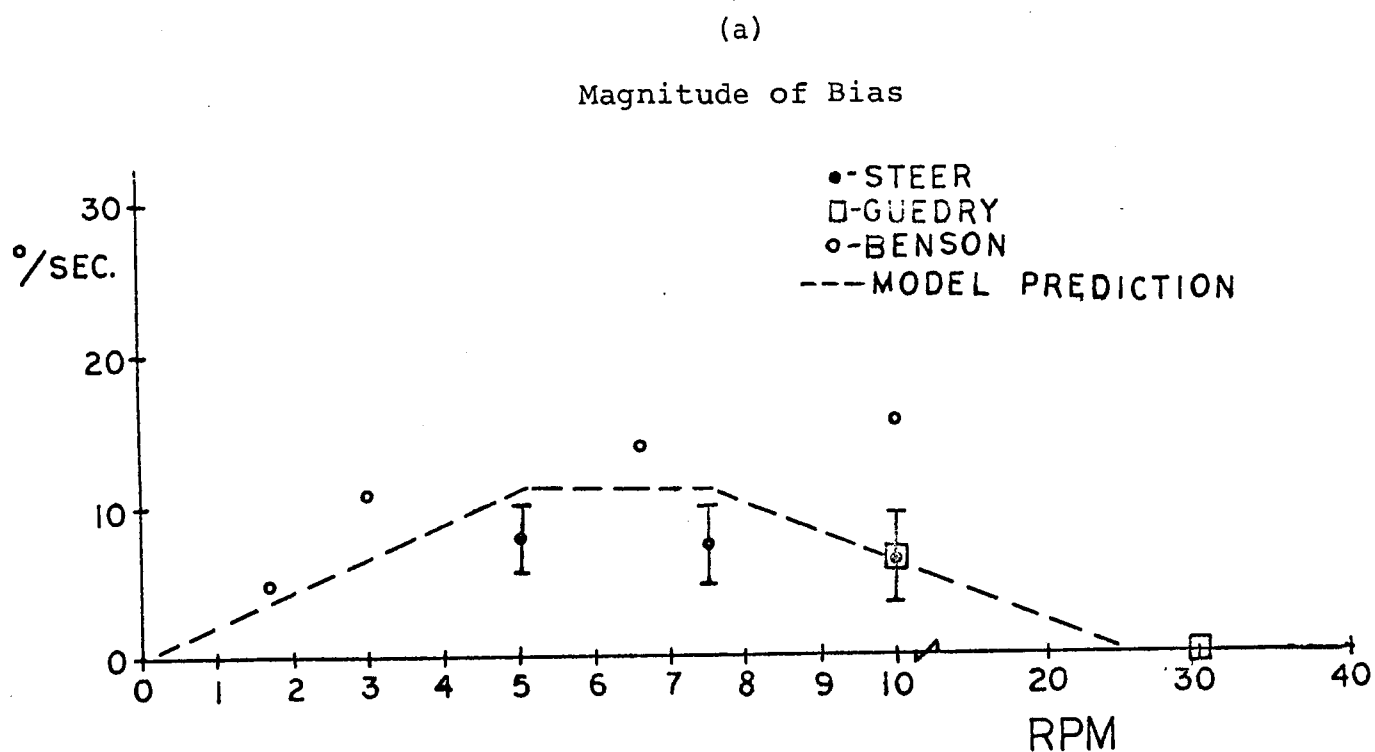


Figure 4

Summary of available data of normalized
bias and sinusoidal amplitude of vestibular nystagmus
from rotation in a 1 g field

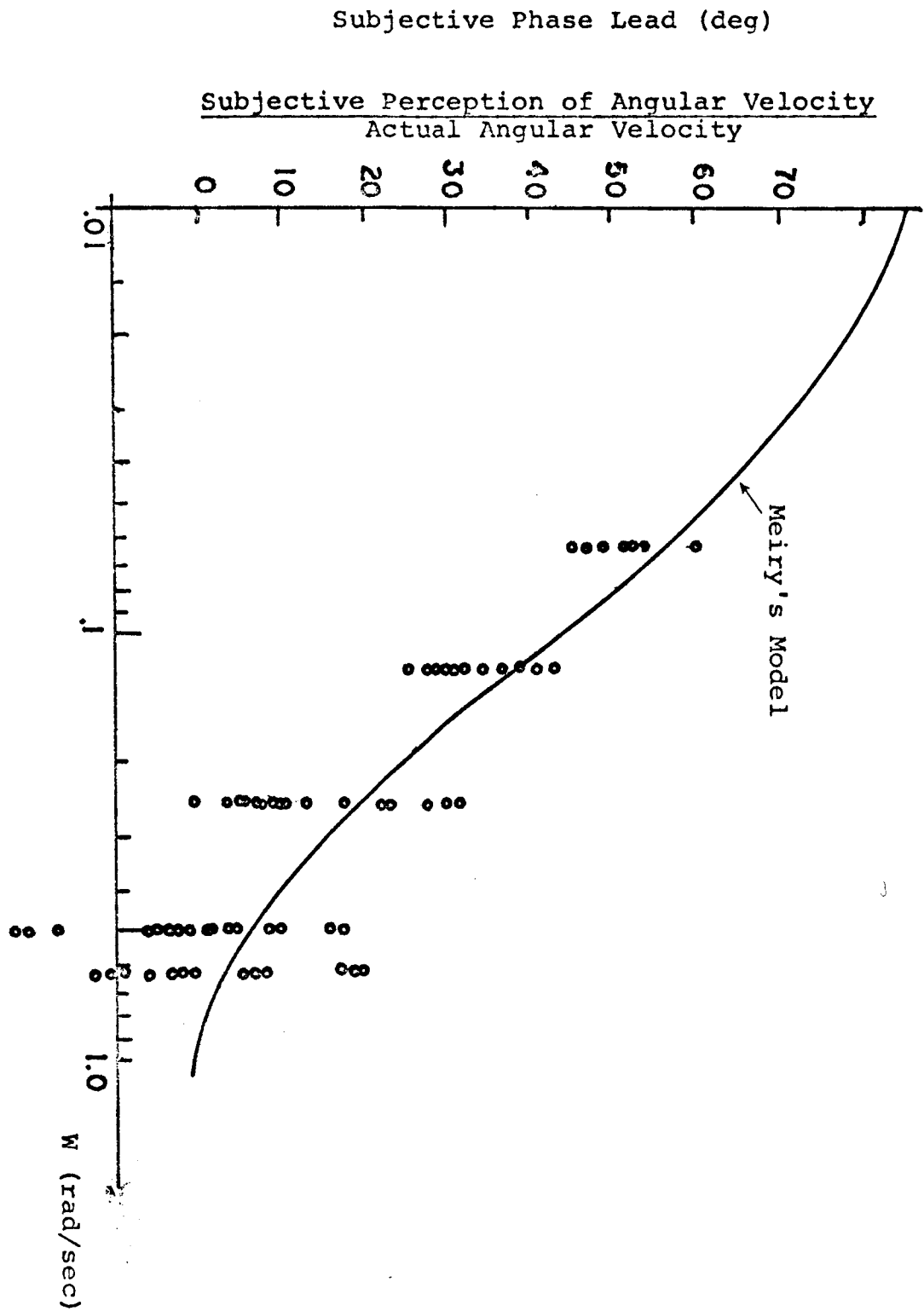


Figure 5. Subjective Measurements

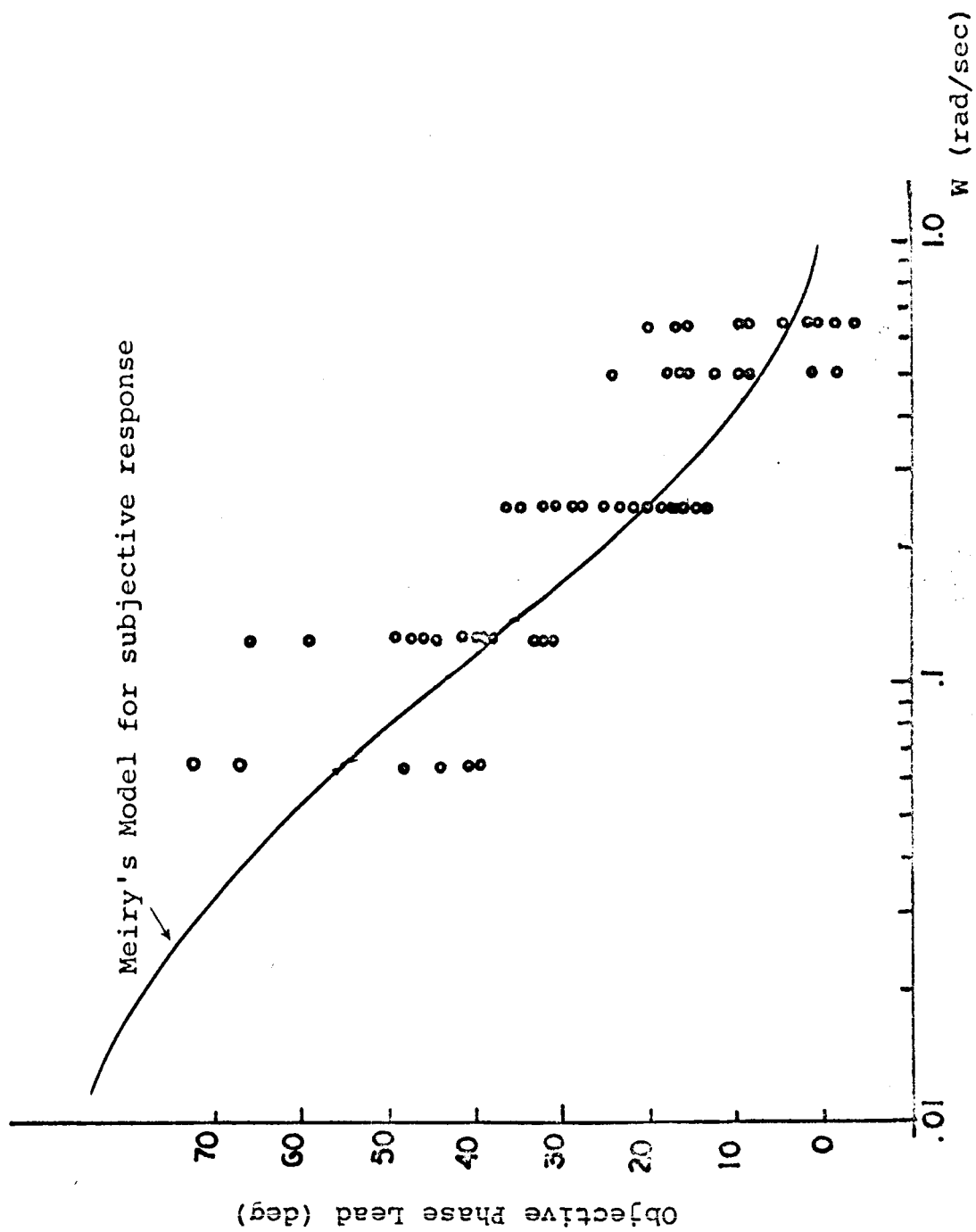


Figure 6. Objective Measurements

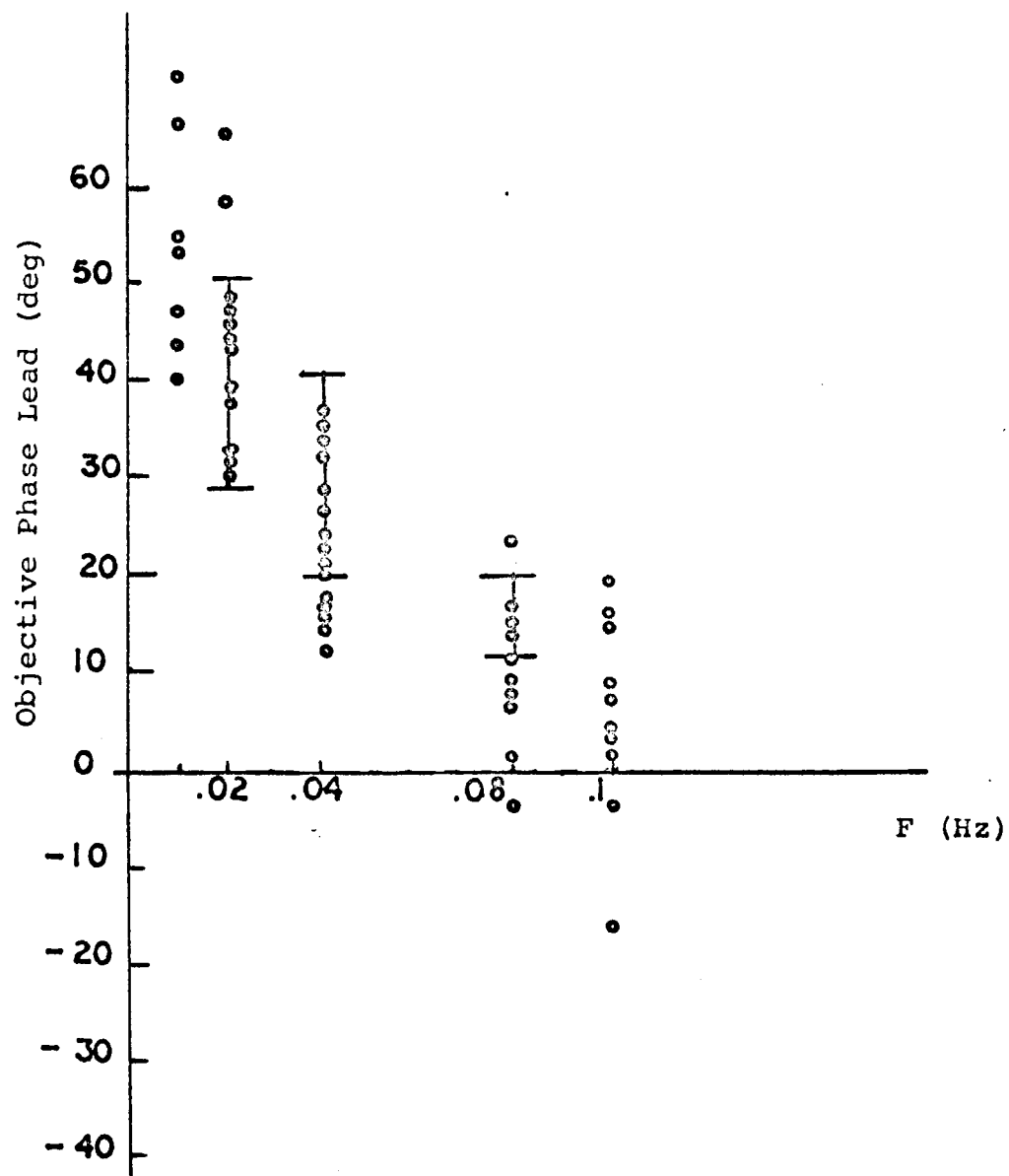


Figure 7
Combined Results of Experiments vs. Hixon and Niven
Combined Results